CHAPTER 48

Congestive Heart Failure

KEY TEACHING POINTS

- In patients with chest pain or dyspnea, the following physical signs all increase the probability of elevated left heart filling pressures and cardiac dyspnea: positive abdominojugular test, abnormal Valsalva response, displaced apical impulse, heart rate greater than 100 beats/minute, the third heart sound, and elevated neck veins.
- A *normal* Valsalva response and *negative* abdominojugular test *decrease* probability of elevated left heart filling pressures.
- The following physical signs are accurate signs of low ejection fraction: Cheyne-Stokes respirations, displaced apical impulse, abnormal Valsalva response, elevated neck veins, and third heart sound.

I. INTRODUCTION

Heart failure is a clinical syndrome characterized by impaired ventricular performance, elevated diastolic filling pressures, and diminished exercise capacity. Patients with heart failure and ventricular disease may have a low ventricular ejection fraction (systolic dysfunction) or normal ejection fraction (diastolic dysfunction).

Clear descriptions of the signs of heart failure date to the Middle Ages.[1] In the 17th century, just after Harvey published his discovery of the circulation of blood, clinicians began to correlate the pathologic observation of large heart chambers and congested lungs with the clinical observations of dyspnea and edema.[2]

II. THE FINDINGS

Many of the findings of heart failure are discussed fully in other chapters of the book, including pulsus alternans and the dicrotic pulse (see Chapter 15), Cheyne-Stokes respirations (see Chapter 19), crackles (see Chapter 30), elevated neck veins (see Chapter 36), the abdominojugular test (see Chapter 36), displaced apical impulse (Chapter 38), and third heart sound (see Chapter 41).

This chapter reviews one finding not discussed extensively elsewhere, the abnormal Valsalva response, and then presents the diagnostic accuracy of all findings of congestive heart failure.

III. THE VALSALVA RESPONSE

A. INTRODUCTION

The **Valsalva maneuver** consists of forced expiration against a closed glottis after a full inspiration.[3] The **Valsalva response** refers to the changes in blood pressure and

pulse that occur during both the strain phase of the maneuver and the recovery period after the strain is released.

Valsalva introduced his maneuver in 1704 as a technique to expel pus from the middle ear.[3-5] The maneuver was forgotten, however, until 1859, when Weber showed he could use it to interrupt his arterial pulse at will (an experiment he eventually abandoned after fainting and developing convulsions).[4] Beginning in the 1950s many different investigators reported that the Valsalva response was distinctly abnormal in patients with congestive heart failure.[6-10]

B. TECHNIQUE

To perform the maneuver, the patient should take a deep breath in and bear down, as if straining to have a bowel movement. The clinician measures the Valsalva response by using a blood pressure cuff, as described later. In clinical studies the straining phase is standardized by having the patient's mouthpiece connected to a pressure transducer, which should demonstrate an increment of 30 to 40 mm Hg for at least 10 seconds.

The Valsalva maneuver is contraindicated in patients with recent eye or central nervous system surgery or hemorrhage. It is also unwise to perform the maneuver in patients with acute coronary ischemia because it may induce arrhythmias, although in patients with chronic ischemic heart disease the maneuver is safe and was once even used to terminate episodes of angina.[11]

C. THE NORMAL VALSALVA RESPONSE

The normal Valsalva response is divided into four phases (Fig. 48.1).[3] In phase 1, the arterial systolic blood pressure rises briefly because increased intrathoracic pressure is transmitted directly to the aorta. In phase 2, blood pressure falls because of reduced venous return during continuing straining. In phase 3, just after release of straining, pressure falls further because of temporary pooling of blood in the pulmonary veins. In phase 4, the arterial pressure overshoots to levels above the control values, primarily because of reflex sympathetic activity induced by previous hypotension. The changes in heart rate are exactly out of phase with the blood pressure: the heart rate increases during phases 2 and 3 and decreases during phase 4.

The clinician identifies these four phases by inflating the blood pressure cuff on the patient's arm 15 mm Hg higher than the patient's resting systolic blood pressure and maintaining this cuff pressure during the straining phase and for 30 seconds afterward, at the same time listening for Korotkoff sounds just as if measuring blood pressure. Korotkoff sounds appear whenever the patient's systolic pressure exceeds the cuff pressure. Therefore, during the normal Valsalva response, Korotkoff sounds appear during phase 1 and phase 4 but are absent during phases 2 and 3.

D. THE ABNORMAL VALSALVA RESPONSE

In patients with congestive heart failure, there are two abnormal Valsalva responses (see Fig. 48.1): (1) **absent phase 4 overshoot**, in which the arterial pressure fails to rise during phase 4 (Korotkoff sounds during phase 1 only), and (2) **square wave response**, in which the arterial pressure rises in parallel with intrathoracic pressure (Korotkoff sounds during phases 1 and 2 only).

In all three interpretable responses—normal, absent phase 4 overshoot, and square wave response—Korotkoff sounds appear during phase 1. If sounds do not appear during this phase, the intrathoracic pressure did not increase to high enough levels during the maneuver, and the test is therefore *not* interpretable.

β-blocker medications may cause a false-positive response, primarily by eliminating the phase 4 overshoot.[12]

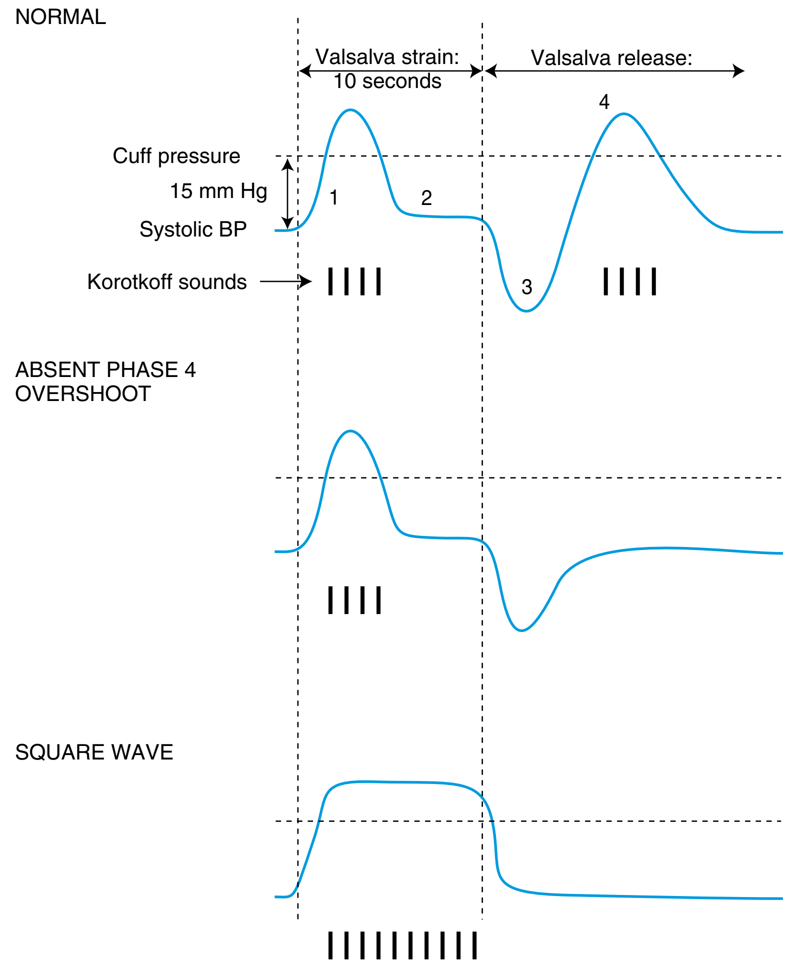

FIG. 48.1 THE VALSALVA RESPONSE. The *solid* line in each drawing depicts changes in systolic blood pressure over time during the Valsalva maneuver. The three types of Valsalva responses are normal (*top*), absent phase 4 overshoot (*middle*), and square wave (*bottom*). The clinician distinguishes these responses by inflating the blood pressure cuff 15 mm Hg above the patient's resting systolic blood pressure (*horizontal dotted line*) and listening for Korotkoff sounds. Korotkoff sounds appear in phase 1 and 4 in the normal response, in phase 1 only in the absent phase 4 overshoot response, and in phases 1 and 2 only in the square wave response. See the text.

E. PATHOGENESIS OF THE ABNORMAL VALSALVA RESPONSE

In patients with congestive heart failure, Korotkoff sounds fail to appear during phase 4 because the weakened heart cannot increase cardiac output in response to hypotension (there is a direct relationship between the degree of overshoot and patient's ejection fraction, $r = 0.72$).[12] Although the cause of the square wave response is still debated, it probably represents the combined effect of neurohormonal activation, peripheral venoconstriction, and increased central blood volume.[8,9,13,14] Phase 2

hypotension may not occur because increased central venous blood volume maintains venous return to the right heart despite the Valsalva strain, and the congested lungs have an ample supply of blood for the left heart.*

IV. CLINICAL SIGNIFICANCE

EBM Boxes 48.1 and 48.2 present the diagnostic accuracy of physical signs for congestive heart failure. EBM Box 48.1 refers to the diagnosis of elevated left heart filling pressures and therefore applies to the diagnosis of systolic or diastolic dysfunction. The ability to accurately detect elevated left heart filling pressures is especially important in patients with dyspnea because elevated pressures implicate the heart as the cause of the patient's symptoms. EBM Box 48.2 refers to the diagnosis of depressed left ventricular ejection fraction and therefore applies only to the diagnosis of systolic dysfunction.

This information should be applied only to patients similar to those enrolled in the studies cited in EBM Boxes 48.1 and 48.2. These patients were all adults presenting to clinicians primarily for evaluation of chest pain or dyspnea. Most had no prior history of congestive heart failure, and many had alternative explanations for dyspnea, such as lung disease.

A. DETECTING ELEVATED LEFT HEART FILLING PRESSURES

In descending order of their likelihood ratios (LRs), the findings *increasing* the probability of elevated filling pressures the most are a positive abdominojugular test (LR = 8; see EBM Box 48.1), abnormal Valsalva response (i.e., either absent phase 4 overshoot or square wave response, LR = 7.6), displaced apical impulse (LR = 5.8), tachycardia (LR = 5.5), third heart sound (LR = 3.9), and elevated venous pressure (LR = 3.9). The findings of a normal Valsalva response (LR = 0.1) and *negative* abdominojugular test (LR = 0.3) *decrease* the probability of elevated left heart filling pressures. The *absence* of tachycardia, elevated venous pressure, displaced apical impulse, or S_3 gallop are all diagnostically unhelpful (LRs not significant).

Because the pulse rate during the Valsalva maneuver is exactly out of phase of the blood pressure changes, the pulse rate should accelerate during phases 2 and 3 of the normal response (i.e., when the systolic blood pressure is falling; see Fig 48.1). In one study the finding of pulse acceleration during Valsalva strain (i.e., increase in rate of 10%, as detected by rhythm strips) *decreased* the probability of elevated filling pressure (LR = 0.2; see EBM Box 48.2).

The presence of crackles, fourth heart sound, or edema does not indicate elevated left heart filling pressures in these patients. Crackles are unhelpful because they are infrequent in chronic heart failure and because many other disorders causing dyspnea also produce crackles. Even so, if the finding of crackles is instead applied only to patients with known cardiomyopathy (e.g., those awaiting cardiac transplantation), they become a more accurate sign of elevated filling pressure, detecting pulmonary capillary wedge pressures of 20 mm Hg or higher with a sensitivity of 15% to 64%, specificity of 82% to 94%, and positive LR of 2.1. The finding is more accurate in this setting probably because other diagnoses causing crackles have already been excluded.[19,34-36]

A small instrument similar to a digital pulse oximeter has been designed that measures and records the pulse pressure during the Valsalva maneuver.[37] This

*The same pathophysiology probably explains the finding of reversed pulsus paradoxus in some patients with congestive heart failure receiving positive pressure ventilation (see Chapter 15).

EBM BOX 48.1

*Congestive Heart Failure—Elevated Left Heart Filling Pressures**

Finding (Reference)†	*Sensitivity (%)*	*Specificity (%)*	**Likelihood Ratio‡ if Finding Is**	
			Present	***Absent***
Vital Signs				
Heart rate >100 beats/min at rest[15]	6	99	**5.5**	NS
Abnormal Valsalva response[16]	95	88	**7.6**	**0.1**
Pulse increase of ≥10% during Valsalva strain[17]	11	54	**0.2**	1.7
Lung Examination				
Crackles[12,15,18,19]	12-23	88-96	NS	NS
Heart Examination				
Elevated jugular venous pressure[12,15,19]	10-58	96-97	**3.9**	NS
Positive abdominojugular test[19-21]	55-84	83-98	**8.0**	**0.3**
Supine apical impulse lateral to MCL[18]	42	93	**5.8**	NS
S_3 gallop[12,15,18,22]	12-37	85-96	**3.9**	0.8
S_4 gallop[12,23]	35-71	50-70	NS	NS
Other Findings				
Edema[12,15]	10	93-96	NS	NS

*Diagnostic standard: For *elevated left heart filling pressures,* pulmonary capillary wedge pressure greater than 12 mm Hg[18] or greater than 15 mm Hg,[16,19-21] or left ventricular end-diastolic pressure greater than 15 mm Hg[12,15,22,23] or greater than 18 mm Hg.[17]

†Definition of findings: For *abnormal Valsalva response*, absent phase 4 overshoot or square wave response (see the text); for *positive abdominojugular test*, sustained rise in jugular venous pressure during 10 to 15 s of midabdominal pressure (see the text).

‡Likelihood ratio (LR) if finding present = positive LR; LR if finding absent = negative LR.

MCL, Midclavicular line; *NS*, not significant.

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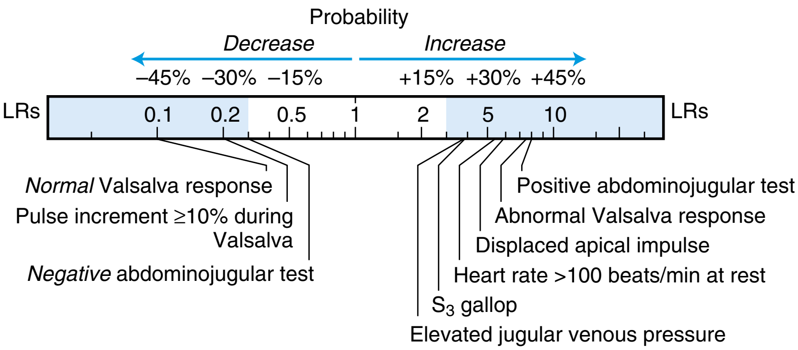
ELEVATED LEFT HEART FILLING PRESSURE



EBM BOX 48.2
*Congestive Heart Failure—Low Ejection Fraction**

Finding (Reference)†	*Sensitivity (%)*	*Specificity (%)*	**Likelihood Ratio‡ if Finding Is** *Present*	*Absent*
Vital Signs				
Heart rate >100 beats/min at rest[24]	22	92	2.8	NS
Cheyne-Stokes Respirations[25]	33	94	**5.4**	0.7
Abnormal Valsalva response[26,27]	69-88	90-91	**7.6**	**0.3**
Lung Examination				
Crackles[24,28-30]	10-29	77-98	NS	NS
Heart Examination				
Elevated neck veins[24,28,30]	7-25	96-98	**6.3**	NS
Supine apical impulse lateral to MCL[24,28-30]	5-66	93-99	**10.3**	0.7
S_3 gallop[22,28,29,31,32]	11-51	85-98	**3.4**	0.7
S_4 gallop[23,33]	31-67	55-68	NS	NS
Murmur of mitral regurgitation[29]	25	89	NS	NS
Other				
Hepatomegaly[28]	3	97	NS	NS
Edema[24,28,30]	8-33	70-98	NS	NS

*Diagnostic standard: For *low ejection fraction*, radionuclide left ventricular ejection fraction less than 0.50[26,27,29,31] or less than 0.53,[28] echocardiographic ejection fraction less than 0.50[22,23,30,32,33] or less than 0.40,[25] or left ventricular fractional shortening less than 25% by echocardiography.[24]
†Definition of findings: For *abnormal Valsalva response*, absent phase 4 overshoot or square wave response (see the text).
‡Likelihood ratio (LR) if finding present = positive LR; LR if finding absent = negative LR.
MCL, Midclavicular line; *NS*, not significant.
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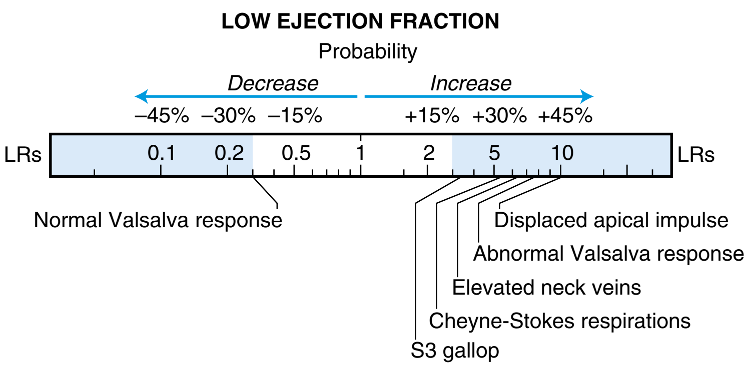

instrument calculates the **pulse-amplitude ratio**, which is the ratio of the pulse pressure at the end of phase 2, divided by that at the beginning of phase 1. Patients with a normal Valsalva response have a low pulse-amplitude ratio (because pulse pressure at the end of phase 2 is much less than that at the beginning of phase 1), whereas those with the square wave response have a higher ratio (near the value of 1). Several studies have shown a direct relationship between the pulse-amplitude ratio and the pulmonary capillary wedge pressure (r = 0.81 to 0.92).[14,37-40] In one study a pulse amplitude ratio of more than 0.7 detected a measured pulmonary capillary wedge pressure of more than 15 mm Hg with a sensitivity of 91%, specificity of 95%, positive LR of 18.2, and negative LR of 0.1,[39] and in another study of elderly patients with heart failure, an elevated pulse amplitude ratio was an independent predictor of mortality.[41]

B. DETECTING DEPRESSED LEFT VENTRICULAR EJECTION FRACTION

Some of the same signs that detect elevated filling pressures also indicate a depressed ejection fraction: displaced apical impulse (LR = 10.3; see EBM Box 48.2), abnormal Valsalva response (either absent phase 4 overshoot or square wave response, LR = 7.6; see EBM Box 48.2), elevated neck veins (LR = 6.3), Cheyne-Stokes respirations (LR = 5.4), and third heart sound (LR = 3.4). Cheyne-Stokes respirations are a more accurate sign of depressed ejection fraction in patients 80 years old or younger (LR = 8.1) than they are in older patients (LR = 2.7) (see Chapter 19).

The *absence* of any of these findings (excepting Valsalva response) is diagnostically unhelpful (i.e., many patients with ejection fractions less than 50% lack these findings). Nonetheless, the absence of the third heart sounds does decrease the probability of an ejection fraction less than 30% (LR = 0.3; see Chapter 41).[29,31]

Some investigators believe that the abnormal Valsalva response is primarily a sign of elevated filling pressure, not low ejection fraction, citing data correlating the degree of Valsalva abnormality with left atrial pressure (r = 0.77, p = 0.005) but not ejection fraction.[16,37,42] This apparent contradiction may reflect varying prevalence of diastolic dysfunction in different investigators' practices. Assuming that the sign is primarily one of elevated filling pressures, it will therefore also be a good sign of depressed ejection fraction if most patients with heart failure in the clinician's practice have systolic dysfunction (see EBM Box 48.2),[26,27] but it will not predict ejection fraction if there is a mixture of patients with systolic and diastolic dysfunction.[16,37,42]

Several findings provide no useful diagnostic information when assessing the patient's ejection fraction: crackles, murmur of mitral regurgitation, hepatomegaly, or edema (all LRs not significant; see EBM Box 48.2).

C. PROPORTIONAL PULSE PRESSURE

In patients with known dilated cardiomyopathy and severe left ventricular dysfunction, a proportional pulse pressure (i.e., arterial pulse pressure divided by the systolic blood pressure) less than 0.25 detects a low cardiac index (i.e., ≤2.2 L/minute per square meter) with a sensitivity of 70% to 91%, specificity of 83% to 93%, positive LR of 6.9, and negative LR of 0.2.[35,43]

D. PHYSICAL SIGNS AND CONSENSUS DIAGNOSIS OF CONGESTIVE HEART FAILURE

Recent investigations[44-52] into the diagnostic accuracy of B-type natriuretic peptide (BNP) in patients with acute dyspnea have further addressed the value of physical examination. In contrast to the studies in EBM Boxes 48.1 and 48.2, these studies

used expert judgment as the diagnostic standard for heart failure, based on the retrospective review of patients' presenting findings, laboratory tests, and response to treatment. These studies confirm the value of the third heart sound (LR = 7.2), displaced apical impulse (LR = 6.7), and elevated neck veins (LR = 4.8); these findings actually increase the probability of heart failure more than a BNP level greater than or equal to 100 pg/mL (LR = 3.6). Nonetheless, in these same studies a BNP level less than 100 pg/mL decreases the probability of the consensus diagnosis of heart failure (LR = 0.1) far more than the *absence* of third heart sound, displaced apical impulse, or elevated neck veins (LRs 0.7 to 0.9).

Because it is possible that judgments about final diagnosis in these studies were influenced by the physical findings themselves, they are excluded from the EBM boxes.

E. PROGNOSIS IN HEART FAILURE

In patients with clinically suspected ischemic heart disease, the physical signs of heart failure are independent predictors of mortality, adding prognostic information to that already provided by the patient's age, exercise capacity, and measured ejection fraction.[53,54] One-year cardiac mortality is higher for those with a displaced apical impulse (39% vs. 12% without the finding, $p = 0.005$), the third heart sound (57% vs. 14% without the finding, $p = 0.002$), and Kussmaul sign (41% vs. 12% without the finding, $p = 0.001$; see Chapter 36).[18,55]

In 1976 Forrester[56] showed that patients with acute myocardial infarction could be classified into four hemodynamic profiles, based on measurements of pulmonary capillary wedge pressure (elevated or not, i.e., *wet* or *dry*) and cardiac output (low or normal, i.e., *cold* or *warm*). Subsequently, clinicians have used physical examination to classify hospitalized patients with heart failure into the same four profiles (i.e., *dry-warm, wet-warm, wet-cold,* or *dry-cold*). In general, *cold* patients have signs of compromised perfusion, such as cool extremities, narrow proportional pulse pressure (<25%; see Chapter 17), pulsus alternans (see Chapter 15), symptomatic hypotension, and impaired mentation. In two studies of 700 heart failure patients, the *cold* profile (either *wet-cold* or *dry-cold*) was associated with increased early mortality (sensitivity 39% to 55%, specificity 83% to 96%, positive LR 5.2).[57,58]

The references for this chapter can be found on www.expertconsult.com.

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